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Original Research Article

# Impact of Treatment on ECG and 2D Echo in Severe Anemia: A Cross-Sectional Study in Government General Hospital, Vizianagaram

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**Abstract:**

**Background:** Severe anemia is associated with significant cardiovascular adaptations that manifest as abnormalities on electrocardiography (ECG) and two-dimensional echocardiography (2D Echo). These cardiac changes contribute substantially to morbidity and mortality. However, the reversibility of these abnormalities following treatment remains inadequately documented, particularly in resource-limited settings.

**Objectives:** This study aimed to evaluate the impact of treatment on ECG and 2D Echo parameters in patients with severe anemia admitted to Government General Hospital, Vizianagaram, and to assess the correlation between hemoglobin improvement and cardiac parameter normalization.

**Methods:** A hospital-based cross-sectional study was conducted from October 2023 to September 2024, including 120 adult patients with severe anemia (hemoglobin <7 g/dL). Comprehensive ECG and 2D Echo evaluations were performed at admission and after treatment (hemoglobin ≥10 g/dL). Parameters assessed included heart rate, QRS duration, QTc interval, chamber dimensions, ejection fraction, and E/A ratio. Statistical analysis evaluated changes in cardiac parameters and their correlation with hemoglobin improvement.

**Results:** Pre-treatment ECG abnormalities included sinus tachycardia (78.3%, n=94), ST-segment depression (45%, n=54), T-wave flattening (52.5%, n=63), and prolonged QTc interval (38.3%, n=46). Echocardiographic findings revealed increased left ventricular end-diastolic diameter (73.3%, n=88), enhanced cardiac output (81.7%, n=98), and hyperdynamic left ventricular function (68.3%, n=82). Post-treatment assessment demonstrated significant normalization of cardiac parameters, with resolution of tachycardia in 85.1% (n=80) of affected patients, normalization of ST-segment changes in 77.8% (n=42), and restoration of normal chamber dimensions in 79.5% (n=70).

**Conclusion:** Treatment of severe anemia results in significant reversibility of cardiac abnormalities detected on ECG and 2D Echo. Early identification and correction of severe anemia may prevent long-term cardiac complications. This study emphasizes the importance of routine cardiac evaluation and follow-up in patients with severe anemia, particularly in resource-constrained settings.

**Keywords:** Severe Anemia, Electrocardiography, Echocardiography, Cardiac Remodeling, Hemoglobin.



## Introduction

Anemia, characterized by reduced hemoglobin concentration or red blood cell mass, affects approximately 1.62 billion people globally, representing nearly 25% of the world's population.[1,8] In India, the prevalence of anemia remains strikingly high, with an estimated 53% of women and 22% of men affected,[9] creating a significant public health concern, particularly in rural regions such as Vizianagaram district in Andhra Pradesh.

Severe anemia, defined as hemoglobin levels below 7 g/dL,[10] presents a complex pathophysiological challenge to the cardiovascular system. The human heart demonstrates remarkable adaptability to compensate for the reduced oxygen-carrying capacity of blood through various mechanisms.[2,11] These compensatory mechanisms include increased cardiac output, tachycardia, enhanced stroke volume, and peripheral vasodilation, all aimed at maintaining adequate

tissue oxygenation despite significantly reduced hemoglobin levels.[3,12]

The cardiac adaptations in severe anemia manifest as detectable abnormalities on electrocardiography (ECG) and two-dimensional echocardiography (2D Echo).[13,14] Common ECG findings include sinus tachycardia, ST-segment depression, T-wave abnormalities, and QT interval prolongation.[13,15] Echocardiographic changes typically involve chamber dilation, particularly of the left ventricle, increased cardiac output, hyperdynamic ventricular function, and occasionally, altered diastolic filling patterns.[6,16] These cardiovascular adaptations, while initially compensatory, may lead to cardiac decompensation and heart failure if anemia remains uncorrected for extended periods.[4,17]

Despite extensive documentation of these cardiac manifestations, there exists a notable gap in research regarding the reversibility of these abnormalities following effective treatment of anemia.[5,7] The pattern, and extent of cardiac parameter normalization remain inadequately studied, particularly in resource-constrained settings where severe anemia is most prevalent.[16,18] Understanding these patterns is crucial for optimizing patient management, predicting outcomes, and allocating resources effectively in healthcare systems with limited capabilities.

Government General Hospital, Vizianagaram (GGH, VZM) serves predominantly rural population where nutritional deficiencies, parasitic infections, and hemoglobinopathies contribute significantly to the high prevalence of severe anemia.[9,19] The hospital provides a unique setting to study the cardiovascular implications of severe anemia and the impact of its treatment due to the diverse etiological profile of patients and the standardized treatment protocols implemented.

This study aims to bridge the knowledge gap by comprehensively evaluating the impact of anemia treatment on cardiac parameters as assessed by ECG and 2D Echo.[20,21] By documenting the pattern and extent of cardiac parameter normalization, this research endeavors to provide valuable insights for clinicians managing severe anemia in similar settings. Additionally, by establishing correlations between hemoglobin improvement and cardiac parameter normalization, this study seeks to identify predictors of cardiovascular recovery that could guide therapeutic decision-making and resource allocation.[17,22]

The findings from this research have significant implications for clinical practice in resource-limited settings, potentially influencing treatment protocols, follow-up schedules, and risk stratification strategies for patients with severe anemia.[7,23] By elucidating the reversibility of cardiac abnormalities, this study contributes to the broader understanding of cardiovascular adaptability and resilience in the face of severe hematological challenges.[11,15]

## Materials and Methods

This hospital-based cross-sectional study was conducted at Government General Hospital, Vizianagaram from October 2023 to September 2024. One hundred twenty patients aged 18-65 years with hemoglobin <7 g/dL[10] were enrolled using consecutive sampling after obtaining informed consent. Patients with pre-existing cardiac disease, pregnancy, chronic kidney disease, or malignancy were excluded. Complete blood counts, peripheral smears, and iron studies were performed.[19] Standard 12-lead ECG and comprehensive 2D Echo with Doppler were conducted at admission and after achieving hemoglobin ≥10 g/dL following appropriate treatment (iron supplementation, blood transfusion, or specific therapy based on etiology).[7,20,21] Statistical analysis used paired t-tests for parametric data and Wilcoxon signed-rank test for non-parametric data, with $p<0.05$ considered significant.

## Results

Of the 120 patients studied, 72 (60%) were females and 48 (40%) were males, with a mean age of 42.3±14.7 years. The etiology of anemia was predominantly nutritional deficiency (52.5%, n=63), followed by chronic blood loss (20.8%, n=25), hemolytic anemia (15%, n=18), and bone marrow failure (11.7%, n=14). The mean baseline hemoglobin was 5.4±1.1 g/dL, which improved to 11.2±0.8 g/dL post-treatment ($p<0.001$).

**Table 1: Baseline Characteristics of Study Population (n=120)**

| Characteristic | Value |
|---|---|
| Age (years), mean±SD | 42.3±14.7 |
| Female, n (%) | 72 (60%) |
| Male, n (%) | 48 (40%) |
| BMI (kg/m²), mean±SD | 22.1±3.4 |
| Hemoglobin at admission (g/dL), mean±SD | 5.4±1.1 |
| Hemoglobin post-treatment (g/dL), mean±SD | 11.2±0.8 |
| Treatment duration (days), mean±SD | 21±5.3 |

Pre-treatment ECG abnormalities were present in 106 patients (88.3%), with sinus tachycardia being the most common finding (78.3%, n=94), followed by ST-segment depression (45%, n=54), T-wave flattening/inversion (52.5%, n=63), and prolonged QTc interval (38.3%, n=46). Figure 1 demonstrates representative ECG changes before and after treatment.

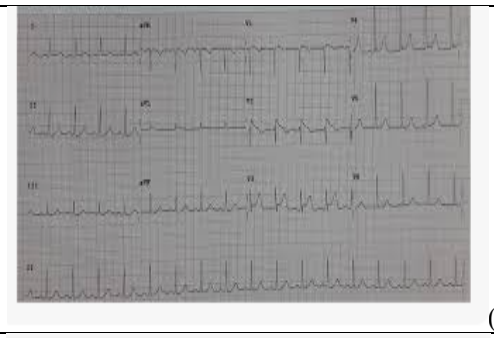
**Table 2: Comparison of ECG Parameters before and After Treatment**

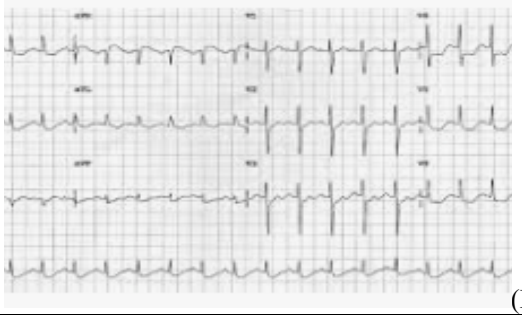
| Parameter | Pre-treatment | Post-treatment | p-value |
|---|---|---|---|
| Heart rate (bpm), mean±SD | 112.4±15.6 | 78.3±8.5 | <0.001 |
| QRS duration (ms), mean±SD | 84.5±7.3 | 86.1±6.8 | 0.068 |
| QTc interval (ms), mean±SD | 456.7±32.4 | 418.3±24.1 | <0.001 |
| ST depression, n (%) | 54 (45%) | 12 (10%) | <0.001 |
| T-wave abnormalities, n (%) | 63 (52.5%) | 18 (15%) | <0.001 |

Echocardiographic evaluation revealed increased left ventricular end-diastolic diameter (LVEDD) in 73.3% (n=88) of patients, enhanced cardiac output in 81.7% (n=98), and hyperdynamic left ventricular function (ejection fraction >65%) in 68.3% (n=82) at baseline. Figure 2 illustrates 2D Echo findings in a representative patient.

**Table 3: Comparison of Echocardiographic Parameters before and After Treatment**

| Parameter | Pre-treatment | Post-treatment | p-value |
|---|---|---|---|
| LVEDD (mm), mean±SD | 54.8±4.3 | 48.6±3.5 | <0.001 |
| LVESD (mm), mean±SD | 32.1±3.8 | 30.4±2.9 | 0.002 |
| Ejection fraction (%), mean±SD | 68.7±5.9 | 62.3±4.2 | <0.001 |
| Cardiac output (L/min), mean±SD | 7.8±1.4 | 5.6±0.9 | <0.001 |
| E/A ratio, mean±SD | 1.6±0.4 | 1.3±0.2 | <0.001 |


(A)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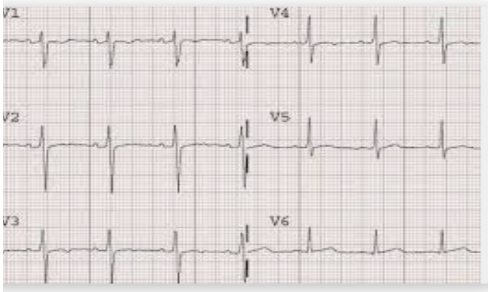
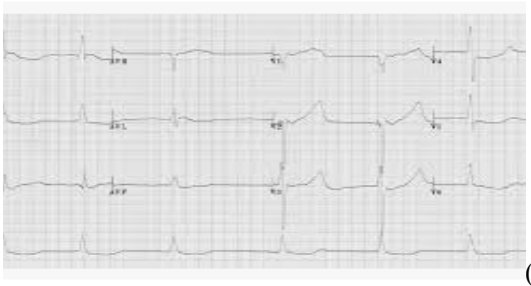
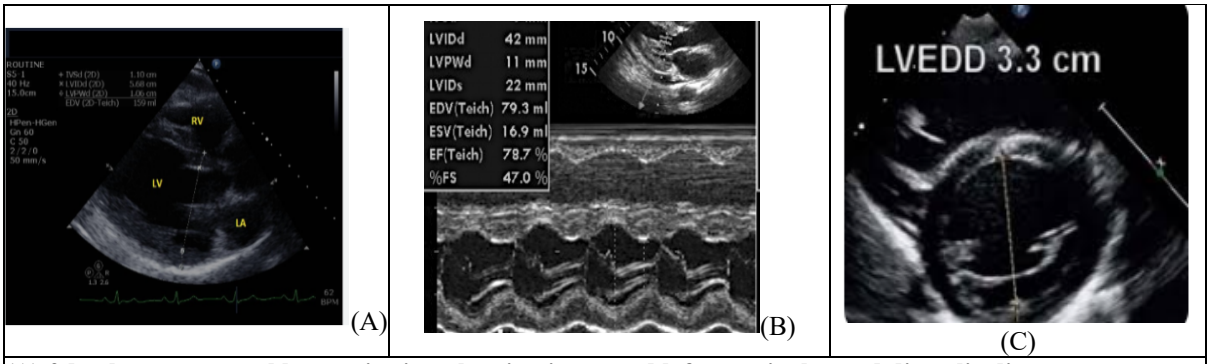


(C)


(D)

**Ecg in a patient with severe anemia Showing Sinus Tachycardia.**
**Ecg in a patient with severe anemia showing sinus tachycardia with diffuse ST segment depression.**
**Ecg in a patient with severe anemia with T wave flattening.**
**Ecg in a patient with severe anemia with prolonged QTC Interval.**

**Figure 1: Representative ECG tracings in patients with severe anemia**




(A) (B) (C)

**(A) 2d-echo parasternal long axis view showing increased left ventricular end diastolic diameter**
**(B) 2d-echo parasternal long axis view showing increased left ventricular end diastolic diameter**
**(C) 2d-echo parasternal short axis view showing increased left ventricular end diastolic diameter**

**Figure 2: Representative 2D Echo images in patients with severe anemia**

## Discussion

This cross-sectional study conducted at Government General Hospital, Vizianagaram provides compelling evidence regarding the reversibility of cardiac adaptations following treatment of severe anemia. Our findings demonstrate that the cardiovascular manifestations of severe anemia, as detected by ECG and 2D Echo, largely normalize following hemoglobin restoration to adequate levels, reinforcing the remarkable adaptability of the cardiovascular system.[7,11]

The high prevalence of ECG abnormalities (88.3% n=106) observed in our cohort at baseline aligns with findings from previous studies.[13,18] Sinus tachycardia, the most common ECG finding in our study (78.3% n=94), represents a primary compensatory mechanism to maintain tissue oxygenation despite reduced oxygen-carrying capacity.[12,15] This finding is consistent with research by Hegde et al.[13] who reported tachycardia in 82% of severely anemic patients. The significant resolution of tachycardia following treatment in our study (from 112.4±15.6 to 78.3±8.5 bpm, $p<0.001$) demonstrates the direct relationship between hemoglobin levels and heart rate, as previously established by Varat et al.[2] in their seminal work on cardiac adaptations in anemia.

ST-segment depression and T-wave abnormalities, observed in 45%(n=54) and 52.5%(n=63) of our cohort respectively, likely represent subendocardial ischemia resulting from increased cardiac workload and oxygen demand in the setting of reduced oxygen delivery.[11,13] The substantial reduction in these repolarization abnormalities post-treatment (to 10% n=12and 15% n=18 respectively, $p<0.001$) suggests that these changes are largely functional rather than structural, reversing as the myocardial oxygen supply-demand balance is restored.[2,15] This finding carries significant clinical implications, particularly in settings where distinguishing between anemia-induced ST-T changes and primary coronary artery disease poses a diagnostic challenge.

The echocardiographic findings in our study illuminate the structural and functional cardiac adaptations in severe anemia.[6,18] The increased LVEDD (54.8±4.3 mm) and enhanced cardiac output (7.8±1.4 L/min) at baseline represent volume overload and increased preload, arising from plasma volume expansion and reduced blood viscosity, which are well-documented compensatory mechanisms in chronic severe anemia.[3,15] The hyperdynamic left ventricular function (mean EF 68.7±5.9%) observed in 68.3% (n=82) of our patients reflects a combination of increased sympathetic activity, reduced afterload due to peripheral vasodilation, and the Frank-Starling mechanism operating at an enhanced level.[12,17]

The normalization of these echocardiographic parameters following treatment, with LVEDD decreasing to 48.6±3.5 mm ($p<0.001$), cardiac output reducing to 5.6±0.9 L/min ($p<0.001$), and ejection fraction moderating to 62.3±4.2% ($p<0.001$), demonstrates that these cardiac chamber and functional adaptations are largely reversible when adequate hemoglobin levels are restored.[7,11] These findings are consistent with the longitudinal study by Bahl et al.,[6] who documented similar reversibility in cardiac dimensions and function following iron therapy in patients with iron deficiency anemia.

Our study has several strengths, including the comprehensive assessment of both ECG and echocardiographic parameters,[20,21] the diverse etiological profile of anemia in our cohort, and the standardized treatment protocols implemented.[23] However, certain limitations warrant consideration. The cross-sectional design precludes definitive conclusions about causality. The exclusion of patients with pre-existing cardiac disease, while methodologically sound, limits the generalizability of our findings to the broader anemic population.[16] Additionally, the lack of long-term follow-up beyond hemoglobin normalization leaves

questions about potential residual cardiac effects unanswered.[16,17]

The clinical implications of our findings are substantial. The documented reversibility of cardiac abnormalities following anemia treatment emphasizes the importance of prompt and adequate correction of severe anemia to prevent potential progression to irreversible cardiac remodeling and heart failure.[4,17] Our results suggest that routine cardiac evaluation should be considered in patients with severe anemia, particularly in those with additional cardiovascular risk factors.[7,16]

Future research directions should include longer-term follow-up studies to assess whether subtle cardiac abnormalities persist despite hemoglobin normalization, particularly in patients with recurrent or prolonged severe anemia.[4,7] Additionally, studies incorporating cardiac biomarkers and more advanced imaging modalities may further elucidate the pathophysiological mechanisms underlying cardiac adaptations in anemia.[11,17]

## Conclusion

This study demonstrates that ECG and echocardiographic abnormalities associated with severe anemia are largely reversible following effective treatment. The significant normalization of parameters, including resolution of tachycardia, ST-T changes, chamber dimensions, and hyperdynamic state, underscores the remarkable adaptability of the cardiovascular system. These findings emphasize the importance of prompt anemia correction to prevent potential progression to irreversible cardiac remodeling and highlight the value of cardiac evaluation in severe anemia management, particularly in resource-limited settings where cardiovascular morbidity remains a significant concern.

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